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DEVELOPMENT OF PROVINCIAL WATER QUALITY GUIDELINES FOR CHLORINATED DIOXINS AND FURANS

BY

MART LUPP AND
LYNN S. McCARTY

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PREAMBLE

The surface water quality management goal for Ontario as stated in "Water Management" (MOE, 1978), is - "to ensure that the surface waters of the province are a quality which is satisfactory for aquatic life and recreation."

In order to achieve this goal, a comprehensive understanding of, and sound management program for the control of physical, chemical and bacteriological pollutants is required. Since 1978 Ontario has relied on Provincial Water Quality Objectives (PWWQOs) and guidelines as principal tools for surface water quality management. The process of developing these has evolved with experience so it is now more effective at taking into account crucial aspects of the hazard of substances in aquatic environments.

Objectives are numerical and narrative values designed for the protection of aquatic life and recreation. All aspects of aquatic life cycles are considered during indefinite exposure to the water. The application of data relating to acute and chronic, lethal and sublethal toxicological effects on aquatic life as well as other phenomena such as bioaccumulation play a major role in establishing these values. Other effects such as taste and odour in water and tainting of fish flesh are also considered.

In recognition that many substances in the aquatic environment lack sufficient data to meet the minimum requirements for the development of Provincial Water Quality Objectives (PWQOs), the Water Resources Branch of the Ontario Ministry of the Environment (MOE) has developed a Provincial Water Quality Guideline (PWQG) setting process. This process has been designed to provide a "rational method for rapid and consistent" development of PWQG where insufficient aquatic toxicity data exist for the derivation of a PWQO. Further, it is designed with the intention of deriving the best possible guideline value to protect aquatic life based on the available information. The guideline value is calculated by applying a safety factor (or "final uncertainty factor") to the lowest water concentration reported to cause a deleterious biological effect. The phenomena of bioaccumulation and aesthetic effects caused by chemical contaminants are also considered in the derivation of a PWQG.

This document provides an overview of the aquatic toxicity of selected polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) as obtained from an extensive manual and computerized literature search. Where the available data permitted, suggested PWQGs have been derived. It should be noted that guidelines, by design, are likely to be over-protective (i.e. more stringent than a PWQO, if sufficient data were available to derive an objective), however, as more data become available, the guidelines will be subject to revision.

EXECUTIVE SUMMARY

Water quality guidelines to protect aquatic life have been developed for 2,3,7,8 - tetrachlorodibenzo-p-dioxin (2,3,7,8-T_4CDD) and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-T_4CDF). The recommended guidelines are:

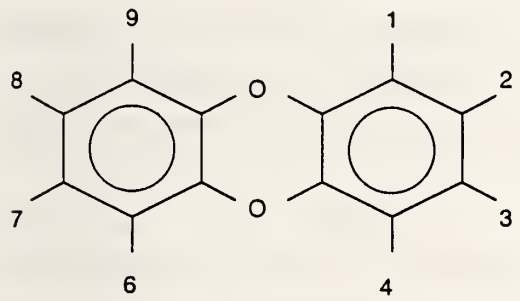
0.1 pg/L for 2,3,7,8-T4CDD, and
0.2 pg/L for 2,3,7,8-T4CDF.

These guidelines are based on an extensive review of the current literature and were derived consistent with the prescribed procedures outlined in the Ministry of the Environment's "Provincial Water Quality Guideline (PWQG) Development Process" document.

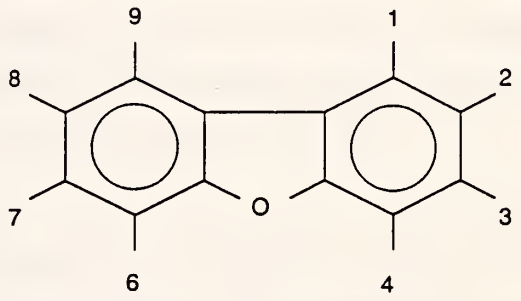
The recommended guidelines are both based on aquatic toxicity considerations: taste and odour and bioaccumulation factors were also considered, but they did not produce values more stringent than the toxicity data. Information on the other chlorinated dioxins and dibenzofurans evaluated in this document were not available to derive PWQG's.

1.0 INTRODUCTION

Polychlorinated dibenzo-p-dioxins (PCDDs) are composed of a triple-ring structure consisting of two benzene rings connected to each other by two oxygen atoms. Depending on the number and position of chlorine substitution on the benzene rings, 75 chlorinated dioxin congeners are possible. The polychlorinated dibenzofuran (PCDF) molecule is also a triple-ring structure with the two benzene rings connected to themselves and by a single oxygen atom (Figure 1). One hundred and thirty-five (135) chlorinated dibenzofuran congeners are possible.



P C D D



P C D F

Figure 1.

PCDDs and PCDFs are often grouped together because they share similar chemical structures, patterns of toxic responses and often common sources to the environment (MOE 1985; EPS 1985). While these classes of compounds are large, most scientific studies have focussed on the most toxic isomer, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-T_4CDD). The general public often erroneously assumes 2,3,7,8-T_4CDD whenever the term dioxin is used.

Sources and releases to the environment have been well documented in the literature (MOE 1985; Hutzinger et al. 1985; EPS 1985; EPA 1985; NRCC 1981; NRCC 1984). PCDD's and PCDF's are not produced intentionally but are unavoidable by-products of chemical manufacturing or the result of incomplete combustion of materials containing chlorine atoms and organic compounds (MOE 1985). PCDDs and PCDFs may also be formed during the disinfection of complex effluents (e.g. pulp and paper) containing many organic constituents.

PCDDs and PCDFs may enter the aquatic environment by the production, use and disposal of chlorophenols and their derivatives (e.g. 2,4-D, 2,4,5-T and hexachlorophene) and atmospheric deposition from combustion sources including municipal incineration, automobiles, coal and wood fired stoves, cigarette smoke and forest fires.

Physical and chemical properties of selected PCDDs and PCDFs are given in Table A in the Appendix. Generally, the environmental distribution and effects of PCDDs and PCDFs are still poorly understood (Baumann and Whittle 1988).

PCDDs are very insoluble in water (i.e. high octanol-water partition coefficients), and are moderately stable exhibiting a relative inertness to acids, bases, oxidation, reduction and heat (Eisler 1986). Although PCDD's are highly persistent, volatilization and photolysis are major removal processes from surface waters. Microbial degradation would appear to be slow (NRCC 1981).

Less is known of the environmental fate of PCDFs, however, just as with PCDDs, they are more likely to be associated with suspended material or found in sediment than dissolved in the water column. For example, Servos (1988) demonstrated that in 40 m^3 lake enclosures less than 15% of the spiked 1,3,6,8-T_4CDD (which has a similar water solubility to 2,3,7,8-T_4CDD) was present in solution while > 85% was sorbed to particulate or dissolved organic matter. The hydrophobicity of PCDD and PCDFs is further illustrated by the fact that they are not detected in open waters of the Great Lakes (detection limit 1 pg/L), although they are detected in fish and sediments from certain locations at ng/kg levels (Muir 1988). In sediment, PCDD and PCDF half-life values are

expected to be 1-2 years, however some evidence suggests these compounds may persist in lake sediments for decades (MOE 1985).

The incidence and levels of PCDDs and PCDFs in fish and other aquatic life have been investigated and/or reviewed by many investigators (MOE 1985; NRCC 1981 and 1984; Baumann and Whittle 1988; Crunkilton et al. 1987; Eisler 1986; Fehringer et al. 1985; Rappe et al. 1987; Ryan et al. 1984 and 1986; Stalling et al. 1983 and 1985; Suns et al. 1983; and Van den Berg et al. 1987).

Generally, PCDFs and PCDDs occur in fish from all the Great Lakes with the highest levels found in fish caught in Saginaw Bay, State of Michigan (Lake Huron). The data indicate low total background levels (1-300 ppt) of a series of PCDDs and PCDFs (Rappe et al. 1987). The toxic 2,3,7,8-substituted PCDDs and PCDFs are usually present in most fish samples. In some cases, 2,3,7,8-T_4CDD has constituted the majority of the total PCDD body burden in fish (Eisler 1986). Detectable levels of 2,3,7,8-T_4CDD typically range between 5 and 20 ppt with the occasional higher value (Ryan et al. 1986). Concentrations are greatest in areas near hazardous waste sites or where chlorinated organic chemicals are manufactured (Stalling et al. 1985).

2.0 TOXICITY TO AQUATIC ORGANISMS

Toxicity data are summarized in Table B.1 and plotted on Forms C in the Appendices. The majority of the available aquatic toxicity literature is for 2,3,7,8-T_4CDD with only limited data available for 2,3,7,8-T_4CDF. Additional aquatic toxicity data which has been called "ancillary" is also appended (Table B.2). These are data which can neither be termed primary nor secondary, and hence cannot be used in the derivation of the final uncertainty factor. For example, this includes studies where oral or intraperitoneal administration of the compounds were used, or studies where no effects were observed at any of the aqueous exposure concentrations tested. The ancillary data is included as it further supports the data used in the guideline derivation process and characterizes the toxic nature of PCDDs and PCDFs.

With 2,3,7,8-T_4CDD the chemical toxicity is manifested long after the initial exposure. Hence, the normal, 96-hour acute toxicity tests are often followed by long observation periods where chronic effects and eventually lethality are observed.

2.1 Acute Toxicity

2.1.1 Fish

Adams et al. (1986) exposed fathead minnows (Pimephales promelas) for 1,2,3 and 4 days to 0.12, 0.72, 7.14 and 81.8 ng/L of 2,3,7,8-T_4CDD. Fish were observed until death or for 150 days. Concentrations as low as 7.1 ng/L combined with exposures of 1 to 4 days produced significant mortality over a post-exposure period of several weeks duration.

Miller et al. (1973 and 1979) investigated the toxicity of 2,3,7,8-T_4CDD to young coho salmon (Oncorhynchus kisutch) exposed for 1 to 4 days and observed for up to an additional 114 days. Two groups of fish with initial mean wet weights of 3.5 and 6.6 g were used. At 5.6 ng/L food consumption, growth and survival were significantly reduced in the smaller fish 60 days post exposure, but not at 0.56 ng/L or lower. For larger fish, growth and survival were reduced at 10.5 ng/L but not at 1.05 ng/L or lower.

Norris and Miller (1974) exposed guppies (Poecilia reticulata) to nominal water concentrations of 100, 1,000 and 10,000 ng/L for 5 days. The effects of exposure were irreversible and all fish died within 37 days, the smaller fish usually dying first. (There were no mortalities among the control fish). Declining interest in feeding and swimming developed after about one week after initial exposure. Fin necrosis occurred in all treated fish surviving more than 10 days. Miller et al. (1979) in a later study exposed guppies for 24 hours to much lower nominal concentrations of 2,3,7,8-TCDD. After maintaining fish for 69 days, the incidence of fin necrosis was significantly greater at 0.1 ng/L than in controls or in 0.01 ng/L exposed fish or in the control fish.

Kleeman et al. (1988) investigated the species differences in 2,3,7,8-T_4CDD toxicity by single intraperitoneal (ip) injection followed by an 80 day observation period (see ancillary toxicity data summary table). The lethal potency of the compound was greater in carp, yellow perch and bullhead than in largemouth bass, rainbow trout or bluegill. With LD50's (80 days post-treatment) of 3 to 5 ug/kg and 10-16 ug/kg, respectively. Neal et al. (1979) demonstrated that a single 500 ug/kg ip injection in the bullfrog (Rana catesbeiana) had no effect on tadpoles through metamorphosis or adults 35 days post-treatment.

Helder (1980a) tested nominal 2,3,7,8-T_4CDD concentrations of 0.1, 1.0 and 10 ng/L on newly fertilized eggs of pike (Esox lucius L.) using 4-day exposures, and followed their development. There was no significant increase in egg mortality up to 10 ng/L. After hatching and during yolk sac absorption, concentrations as low as 1.0 ng/L significantly increased mortality. In the 0.1 ng/L treated group less than 5% had edemas versus 40% and nearly 100% in the 1.0 and 10.0 ng/L treatment groups, respectively. Incidence of edemas in controls was 0%. Other histopathologic changes included haemorrhages, alterations of blood vessel walls, and liver damage.

2.1.2 Invertebrates

Data on acute exposure of 2,3,7,8-T_4CDD are very limited. Daphnia magna aged < 1, 7 and 21 days were exposed for 48 hours to concentrations ranging from 0.2

to 1030 ng/L and maintained for 9 days. No effects at any of the exposure concentrations were noted (Adams et al. 1986).

There is no information on the acute exposure toxicity of any other chlorinated dioxins or furan isomers to invertebrates.

2.2 Chronic Toxicity

2.2.1 Fish

Yochim et al. (1978) exposed channel catfish (Ictalurus punctatus) fingerlings and mosquito fish (Gambusia affinis) for up to 20 days in a recirculating aquatic model ecosystem. Measured 2,3,7,8-T_4CDD exposure concentrations ranged from 2.4 to 4.2 ng/L and were derived from 0.1 ppm ^{14}C-T_4CDD treated flooded soil. All unharvested fish died after 14 to 20 days of exposure.

Helder (1980b, 1981) exposed the early life stages (e.g. freshly fertilized eggs, yolk sac fry, 0.85 g juveniles) of rainbow trout (Salmo gairdneri) to nominal concentrations of 2,3,7,8-T_4CDD for 4 consecutive days. Exposure of eggs to even 0.1 ng/L induced retardation of embryonic development and growth, and fry growth. At higher concentrations (1.0 -100 ng/L), the compound caused dose related incidences of haemorrhages, generalized edema and liver injury, followed by death. In the juvenile trout, growth retardation, delayed mortality and formation of inclusion bodies in the stomach, pancreas and liver were manifested in the treated fish.

Mehrle et al. (1988) exposed rainbow trout fry to 2,3,7,8-T_4CDD for 28 days followed by a 28 day depuration period. Concentrations in the water were measured every 7 days. At a mean exposure concentration of 0.038 ng/L (lowest concentration tested) fish were obviously stressed as judged by reduced growth and behavioral responses (e.g swimming posture, inhibition of feeding, lack of response to external stimuli). Significant mortality occurred during the depuration phase. The 28 day LC50 was determined to be 0.176 ng/L.

Adams et al. 1986 determined a 28 day exposure LC50 of 1.7 ng/L for fathead minnow (Pimephales promelas) with 2,3,7,8-T_4CDD.

Cook et al. (1986) showed exposure of 0.1 ng/L of 2,3,7,8-T_4CDD for 71 days resulted in weight loss and other chronic effects in carp.

The data for 2,3,7,8-T_4CDF is limited to the work of Mehrle et al. (1988). Rainbow trout were exposed for 28 days and maintained for an additional 28 days. At 0.41 ng/L no observed effects on growth were noted. The LOEC for growth retardation was 0.90 ng/L. At 3.93 and 8.78 ng/L significant mortality was observed within 14 days.

2.2.2 Invertebrates

Data for chronic exposure to invertebrates are only available for 2,3,7,8-T_4CDD. Miller et al. (1973) exposed juvenile and adult snails (Physa sp.), mosquito larvae (Aedes aegypti) and adult (40 mm) oligochaete worms (Paranais sp.) to an initial nominal concentration of 200 ng/L for 36, 17, and 55 days, respectively. No significant effects on adult snail survival or egg production were noted. Snail eggs completed their development in the original exposure solution. After 48 days from the beginning of the experiment, live juvenile snail shells and empty juvenile snail shells were counted. The total snail hatch in the treated groups was lower (approx. 30% but $P = 0.056$), but there was no significant difference in the survival of the juveniles. For the mosquito larvae no significant difference was observed in total pupation or the rate of pupation among treated and control mosquitoes. For the worm, the total number of worms present at the end of the 55 day exposure period were significantly lower and the total worm biomass was reduced indicating 2,3,7,8-T_4CDD exerted its effects primarily on reproduction rather than growth of the individual worms.

Yochim et al. (1978) exposed adult Daphnia magna and adult snails, Helosoma sp. to 2.4 to 4.2 ng/L of 2,3,7,8-T_4CDD for 32 days. No effects on feeding, growth or reproductive activity was observed in either organism.

2.2.3 Aquatic Plants/Algae

Yochim et al. (1978) using recirculating static model ecosystems found 2,3,7,8-T_4CDD had no effect on the alga, Oedogonium cardiacum after 32 days exposure to 2.4 -4.2 ng/L. Similarly, Isensee and Jones (1975) reported no toxicity in their static bioaccumulation studies using O. cardiacum and duckweed (Lemna minor) where 2,3,7,8-T_4CDD concentrations ranged from 0.05 to 1300 ng/L.

No toxic effects were noted on pond weeds, Elodea nuttali and Certophyllum emersum after exposure to 53.7 ng/L for several months (Tsushimoto et al. 1982).

Zullei and Benecke (1978) conducted contact inhibition studies with filamentous algae, Phormidium sp. where filter paper was spotted in 3 places with 1 ug of 2,3,7,8-T_4CDD. Filtered algae on disks were placed on the spots and the filter paper placed in a petri dish containing a nutrient medium. Algal filament motility outward from the disks was measured over 3 hours. A significant inhibition of motility relative to controls was noted. However, extrapolation to a water exposure concentration cannot be made.

2.3 Summary of Toxicity Data

The available toxicity data is primarily for 2,3,7,8-T_4CDD. The data indicates that this dioxin is a slow-acting toxicant with acute exposures of only 1 to 4 days leading to mortality several weeks post-treatment.

The most readily observed sublethal effect to fish is a reduction in growth, along with fin necrosis, edema and behavioral changes.

It appears that embryonic and larval life stages of fish are the most sensitive aquatic life forms. Based on the accumulated toxicity data in the appendix, including ip and oral feeding studies, concentrations in whole fish greater than 1000 ng/kg of 2,3,7,8-T_4CDD are associated with sublethal effects such as reduced growth (Muir 1988).

The species of snails, insects, cladocerans and worms tested seem to be considerably less sensitive than fish. Aquatic plants and algae appear to be relatively insensitive to 2,3,7,8-T_4CDD.

The rainbow trout data of Mehrle et al. (1988) indicates that 2,3,7,8-T_4CDF is approximately a factor of 10 times less toxic than 2,3,7,8-T_4CDD in the case of growth effects. This ratio is consistent with mammalian toxicology literature (Muir 1988).

3.0 BIOACCUMULATION/BIOCONCENTRATION

Bioaccumulation/bioconcentration of PCDDs and PCDFs have been reviewed by several authors (MOE 1985; EPA 1985; Kenaga and Norris 1983; and Muir 1988). Experimental data are based on static test chambers under a number of experimental protocols which are not necessarily comparable. These include studies of uptake kinetics based on water, sediment and dietary exposures. The accumulated data highlight the metabolic and bioavailability differences (variability) among the isomers and aids in interpreting the toxicological information and environmental fate of these compounds. The data further illustrates the problem of attempting to address the dioxins and furans as a group.

The accumulation of PCDDs and PCDFs by aquatic biota is congener-and isomer-specific. It appears that there is a preferential accumulation of isomers with the 2,3,7, and 8 positions filled with chlorine and in particular, 2,3,7,8-T_4CDD. (Adams et al. 1986; Stalling et al. 1983; Niimi and Oliver 1986; Kuehl et al. 1986, 1987a and 1987b; and Van den Berg et al. 1987).

Experimentally determined whole organism BCF (wet weight basis) for 2,3,7,8-T_4CDD are usually in the range of 2000-15500 based on fish, algae, snail and daphnia investigations. (BCF have been calculated as either the ratio of tissue concentration to water exposure concentration, or uptake rate to depuration rate). The experimentally determined BCFs are much less than predicted on the basis of solubility models where estimates can exceed 100,000. (Adams et al. 1986; EPA 1985; Servos 1988).

There appear to be four primary causes for the differential accumulation of isomers and inability for conventional models to predict the BCF of dioxin and furan isomers in the laboratory.

i) Differences in bioavailability. Water solubility over the range of isomers varies dramatically and especially with the higher chlorinated isomers the chemical is not available to the typically examined targeted organism directly, but rather sediment and contaminated food appear to be the primary exposure pathway (Gobas et al. 1986; Kuehl et al. 1987a; Muir

et al. 1988; Servos 1988). Also humic acids and other organic material in the water column can substantially influence bioavailability. (Webster et al. 1986; Servos 1988).

ii) Isomer-specific variations in uptake and depuration rates. The fact that there are isomer-specific (and congener-specific) differences in uptake and elimination rates, both from food and water, is well documented (Muir et al. 1985a, 1985b, 1986, 1988; Niimi and Oliver 1986; Gobas et al. 1986; Sijm and Opperhuizen 1988).

Where uptake is primarily by diet, not only are rates different than water-borne uptake rates, but complications related to variable feeding rates and assimilation efficiencies further confound the issue. Sijm and Opperhuizen (1988) showed that 2,8-D_2CDD was rapidly metabolized by goldfish (Carassius auratus) while 2,3,7,8-T_4CDD was not. When an inhibitor of 2,8-D_2CDD metabolism (piperonylbutoxide) was used, the 2,8-D_2CDD accumulated in the fish and exhibited a toxicity 60 times less than 2,3,7,8-T_4CDD. In the absence of the metabolic inhibitor, the toxicity of 2,8-D_2CDD was not measurable.

Molecular size may also have a bearing on rate and extent of uptake in short exposures. It is proposed that compounds having cross-sections greater than 0.95 nm are too large for passive membrane transport. This, for example, would reduce bioaccumulation of O_8CDD and 1,4 and 1,6 chlorine substituted dioxins (Gobas et al. 1986; Sijm and Opperhuizen 1988).

iii) Species differences. Even for a specific isomer, there is evidence to indicate that substantial differences in metabolic degradation rates and pathways exist between different species. Kleeman et al. (1988) showed that for the six fish species injected ip, species differences existed in the biotransformation of 2,3,7,8-T_4CDD as well as species differences in the signs of overt toxicity and lethal potency.

iv) Exposure time. Exposure time may have been too short for an organism-water equilibrium steady-state to be reached.

Monitoring results indicate that highly chlorinated dioxins in carp, a detrital feeder, do not accumulate to levels measured in sediment (Stalling et al. 1983). The uptake of TCDD from sediment is the subject of an extensive bioaccumulation study currently underway by the U.S. EPA and N.Y. State Departments of Environmental Conservation and Health (EPA 1988).

Only 2,3,7,8-substituted T_4CDDs and P_5CDDs, and T_4CDFs to H_6CDFs, appear to biomagnify based on monitoring data (Muir 1988).

BCF data for selected PCDDs and PCDFs are provided in the following table. Of the reported BCFs, 5,000 was selected as the "best current estimate" for 2, 3, 7, 8 - T_4CDD by the U.S. EPA (EPA 1985).

TABLE 1

BIOACCUMULATION FACTORS FOR AQUATIC ORGANISM

Compound (Reference)	Algae/Plants	Snails	Daphnids (*Daphnia magna*)	Mosquito Fish (*Gambusia affinis*)	Catfish (*Ictalurus punctatus*)	Rainbow Trout (*Salmo gairdneri*)	Fathead Minnows (*Pimephales promelas*)	Guppy (*Poecillia reticulata*)
2,3,7,8-T_4CDD (Isensee 1978)	690-6400	390-13000	1180-7250	240-15500	490-6800	-	-	-
2,3,7,8-T_4CDD (Tsushimoto *et al*. 1982)	2000	-	-	-	-	-	2500	-
2,3,7,8-T_4CDD (Yochim *et al*. 1978)	2083	3731	7125	4875	-	-	-	-
2,3,7,8-T_4CDD (Mehrle *et al*. 1988)	-	-	-	-	-	26707	-	-
2,3,7,8-T_4CDF (Mehrle *et al*. (1988)	-	-	-	-	-	2455-6049	-	-
1,2,3,7-T_4CDD (Muir *et al*. 1985b)	-	-	-	-	-	874, 1577	2018, 2458	-
1,2,3,4,7-P_5CDD (Muir *et al*. 1985b)	-	-	-	-	-	810	1647, 1220	-
1,2,3,4,7,8-H_6CDD (Muir *et al*. 1985b)	-	-	-	-	-	1715, 2840	2630, 5834	-

TABLE 1 (CONTINUED)

Compound (Reference)	Algae/Plants	Snails	Daphnids (Daphnia magna)	Mosquito Fish (Gambusia affinis)	Catfish (letalunis punctatus)	Rainbow Trout (Salmo gairdneri)	Fathead Minnows (Pimephales pronelas)	Guppies (Poecillia reticulata)
1,2,3,4,6,7,8-H_7CDD (Muir et al. 1985b)	-	-	-	-	-	1059, 1790	513, 515	-
1,3,6,8-T_4CDD (Muir et al. 1986)	-	-	-	-	-	2938, 1964, 1400	5840, 5565	-
O_8CDD (Muir et al. (1986)	-	-	-	-	-	34, 136	2226	-
2,3,7,8-T_4CDD (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	13000
2,3,7,8-T_4CDF (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	6600
1,2,3,7,8-P_5CDD (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	7700
1,2,3,7,9-P_5CDD (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	1600
1,2,3,7,8-P_5CDF (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	2400
2,3,4,7,8-P_5CDD (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	5000
1,2,3,7,8,9-H_6CDD (Opperhuizen et al. 1986)	-	-	-	-	-	-	-	900

4.0 IMPACTS ON TASTE AND ODOUR OF WATER AND FISH TISSUES

No evidence was found in the literature which would suggest that the presence of dibenzodioxins and dibenzofurans compounds in water would result in any aesthetic impairment. Therefore taste and odour considerations would not be a factor in the guideline development.

5.0 DERIVATION OF PWQGs

Adequate aquatic toxicity information on only 2,3,7,8-T_4CDD and 2,3,7,8-T_4CDF is available for the derivation of PWQGs.

5.1 Calculation of Final Uncertainty Factor

As indicated in Table A of the Appendix, the log kow values for both 2,3,7,8-substituted T_4CDD and T_4CDF are both in excess of 4.0. Therefore baseline uncertainty factors of 10000 have been selected.

The rationale for the selection of calibration factors for toxicity data which qualify for decreasing the baseline uncertainty factor is as follows:

For any data to be classified as primary, static/renewal exposures with measured water concentrations were the necessary minimum pre-requisites. The acceptance of static/renewal procedures for these hydrophobic compounds was a concession in recognition of the potential hazard posed to laboratory workers from long-term flow-through procedures.

Any data from acute exposures of 5 days or less regardless of the length of the subsequent observation period or whether lethal or non-lethal responses were reported were classified as acute data. The only exception to this was the work of Helder (1980b, 1981) where various rainbow trout life stages from eggs to juveniles, were separately exposed for 4 days and observed for up to 72 days.

Form D in the Appendix outlines the derivation of the final uncertainty factors. For 2,3,7,8-T_4CDD the calculation was based on the baseline uncertainty factor of 10000 multiplied by 7 calibration factors yielding a final uncertainty factor of 320. For 2,3,7,8-T_4CDF a final uncertainty factor of 4000 was calculated as the product of the baseline factor of 10000 and 1 calibration factor.

5.2 Calculation of the Guideline Values

2,3,7,8-T_4CDD

For 2,3,7,8-T_4CDD the lowest observed toxic effect concentration was 0.038 ng/L for rainbow trout growth. Dividing by the final uncertainty factor of 320 yields a preliminary PWQG of 0.00012 ng/L or 0.1 pg/L.

In Canada the current acceptable level of 2,3,7,8-T_4CDD in fish (edible portion) is 20 parts per trillion (ppt) based on a consumptive rate of 113 g per week and a safety factor of 218 applied to the NOEL determined in long term animal studies. (Interdepartmental Committee on Toxic Chemicals 1983). The U.S. EPA "best current estimate" of the BCF of 2,3,7,8-T_4CDD for fish is 5000 (EPA 1985). Dividing the 20 ppt fish guideline by 5000 yields the concentration in water of 0.004 ng/L or 4 pg/L to which the fish would have to be exposed to accumulate this level (dietary routes excluded). The application of an additional safety factor of two further reduces this value to 2 pg/L or 20 times the previously determined preliminary guideline for 2,3,7,8-T_4CDD. New York State Department of Environmental Conservation has developed fish flesh criteria to protect piscivorous wildlife (Newell et al. 1987). For 2,3,7,8-T_4CDD a non-carcinogenic based criterion of 0.000003 mg/kg diet (3 ppt) was developed to protect wildlife consuming contaminated fish flesh. Dividing this criterion by the BCF value of 5000 yields a corresponding water concentration of 0.6 pg/L. The application of an additional safety factor of two reduces this concentration to 0.3 pg/L or 3 times the previously determined preliminary guideline.

Therefore the guideline of 0.1 pg/L based on aquatic toxicity considerations is recommended as the PWQG.

2378-T_4CDF

For 2,3,7,8-T_4CDF the lowest reported toxic effect concentration was 0.90 ng/L for rainbow trout growth. Dividing by the final uncertainty factor of 4000 yields a preliminary PWQG of 0.0002 ng/L (0.2 pg/L).

The guideline of 0.2 pg/L for 2,3,7,8-T_4CDF is recommended as the PWQG. The guidelines are shown graphically on Form C in the Appendix.

6.0 RESEARCH NEEDS

It is not known whether residues of 3 ppt or less of 2,3,7,8-T_4CDD in fish represent an unacceptable risk to predators. In the work by Newell et al. (1987) the 3 ppt criterion was based on toxicity to non-target laboratory animals. There is evidence of biomagnification for some isomers and 2,3,7,8-T_4CDD has been associated with poor reproduction success of herring gulls (Larus argentatus) in Lake Ontario (Stolzenburg and Sullivan 1983).

With the exception of 2,3,7,8-T_4CDD, aquatic toxicity data on the other PCDDs and PCDFs are essentially lacking.

Additional testing using long term exposures to pg/L concentrations to a variety of aquatic organisms (vetebrates, invertebrates and plants) is recommended with particular emphasis on the investigation of sublethal effects. Of particular interest to the Ministry of the Environment is the development of an aquatic toxicological data base for dioxins and furans that satisfy the minimum requirements needed for the establishment of Provincial Water Quality Objectives.

Based on the hydrophobicity and importance of dietary uptake, ecosystem model approaches (e.g. aquatic plants and sediments also present with test organisms) which simulate real world exposure conditions are required for improved general understanding of the toxic action of these chemicals.

The toxic effects of some 2,3,7,8,-substituted PCDDs and PCDFs have been shown to be additive if administered in a mixture based on E.L.S. bioassays with rainbow trout (Bol et al. 1988). Mammalian systems have shown the toxicity of mixtures of dioxins and furans can be both additive and non-additive. This may have to be addressed as further guidelines are developed given the fact that environmental exposures are invariably to multiple isomers. For example, while the individual guidelines for both 2,3,7,8-T_4CDF and T_4CDD may be met at a given location, aquatic life may still be stressed due to potential additive effects.

7.0 OBJECTIVES OF THE OTHER AGENCIES

At present there are no numerical criteria or standards promulgated for any PCDD or PCDF by any regulatory agency for the protection of the most sensitive aquatic life form from exposure to the chemicals from water.

The International Joint Commission recommends that for the protection of all life forms, 2,3,7,8-T_4CDD should be absent (not detectable) from all compartments of the ecosystem , including air, land, water, sediment and biota(see * below)(IJC 1980).

The U.S. EPA has developed ambient water quality criteria for the protection of humans exposed to 2,3,7,8-T_4CDD from the consumption of fish and water from the same surface water body (EPA 1984). The recommended criteria are 1.3×10^{-7}, 1.3×10^{-8} and 1.3×10^{-9} ug/L for potential increase in risk of cancer over the lifetime of 10^{-5}, 10^{-6} and 10^{-7} respectively. These criteria assume a daily consumption of 6.5g contaminated fish and shellfish with the daily consumption of 2L of contaminated drinking water.

The Canadian acceptable 2,3,7,8-T_4CDD levels in fish for human consumption and the New York State 2,3,7,8-T_4CDD levels in fish for wildlife consumption were addressed in Section 5.2.

*Note 1: Absent means not detectable as determined by the best available technology.

Note 2: The present (1980) detection limit for TCDD is 0.01 ug/kg in tissue and in sediment and is 0.00001 ug/L in water (10pg/l).

Note 3: Other tetrachlorodioxin isomers and higher chlorinated dioxin congeners are of concern in the Great Lakes Ecosystem. However, the data base is inadequate to support a scientifically defensible recommendation at this time.

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appendix a

TABLE A - PHYSICAL-CHEMICAL PROPERTIES OF SELECTED DIBENZODIOXINS AND DIBENZOFURANS

Compound	CAS No.	Formula	M.W. (g/mol)	Molar Volume (cm^3/mol)	Melting Point (^oC)	Vapour Pressure (Pa)	Water Solub. (mol/m^3)	Log Kow (measured)
2,3,7,8-T_4CDD	1746-01-6	$C_{12}H_4Cl_4O_2$	322	275.6	305	$(4.5\text{-}0.098) \times 10^{-6}$	2.46×10^{-8}	7.02, 6.80
1,2,3,4-T_4CDD	30746-58-8	$C_{12}H_4Cl_4O_2$	322	275.6	190	6.4×10^{-6}	$(1.5\text{-}2.0) \times 10^{-6}$	6.60, 7.18
1,2,3,7,8-P_5CDD	40321-76-4	$C_{12}H_3Cl_5O_2$	357	296.5	240	-	-	7.80
1,2,3,6,7,8-H_6CDD	57653-85-7	$C_{12}H_2Cl_6O_2$	391	317.4	285	-	-	7.62 (non-isomer specific estimate)
1,2,3,7,8,9-H_6CDD	19408-74-3	$C_{12}H_2Cl_6O_2$	391	317.4	243	-	-	7.62 (non-isomer specific estimate)
1,2,3,4,7,8-H_6CDD	39227-28-6	$C_{12}H_2Cl_6O_2$	391	317.4	273	5.1×10^{-9}	1.13×10^{-8}, 1.46×10^{-8}	7.80, 10.22
2,3,7,8-T_4CDF	51207-31-9	$C_{12}H_4Cl_4O$	306	-	-	-	-	5.82
2,3,4,7,8-P_5CDF	57117-31-4	$C_{12}H_3Cl_5O$	340	-	-	-	-	-
1,2,3,7,8-P_5CDF	57117-41-6	$C_{12}H_3Cl_5O$	340	-	-	-	-	-
1,2,3,6,7,8-H_6CDF	57117-44-9	$C_{12}H_2Cl_6O$	375	-	-	-	-	-

TABLE A - CONTINUED

Compound	CAS No.	Formula	M.W. (g/mol)	Molar Volume (cm^3/mol)	Melting Point (^oC)	Vapour Pressure (Pa)	Water Solub. (mol/m^3)	Log Kow (measured)
1,2,3,7,8,9-H_6CDF	72918-21-9	$C_{12}H_2Cl_6O$	375	-	-	-	-	-
2,3,4,6,7,8-H_6CDF	60851-34-5	$C_{12}H_2Cl_6O$	375	-	-	-	-	-
1,2,3,4,7,8-H_6CDF	70648-26-9	$C_{12}H_2Cl_6O$	375	-	-	-	-	-

Sources: MOE 1985; Shiu et al. 1988; EPA 1985; Hutzinger et al. 1985; Eisler 1986; Adams and Blaine 1986; Webster et al. 1986; and the LOGP computerized database (Technical Database Services).

appendix b

TABLE B.1 - PRIMARY AND SECONDARY AQUATIC TOXICITY DATA

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	Test Conditions pH	Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish	water	2.4 - 4.2	15-20	5900 (max.)						100% mortality (water conc'n derived from 0.1 ppm 14C-T_4CDD treated soil).	C/S/M secondary	Yochim et al. 1978
Channel catfish, Ictalurus punctatus (fingerlings)												
Mosquito fish, Gambusia affinis	water	2.4 - 4.2	15	11700 (max.)						100% mortality (water conc'n derived from 0.1 ppm 14C -T_4CDD treated soil).	C/S/M secondary	Yochim et al. 1978 Isensee 1978
Coho salmon, Oncorhynchus kisutch												
(3.5 g)	water	5.6	4	NG	6.9	12-18			64	LC50 (reduced growth food consumption, survival) (LT50 = 60 days)	A/S/U secondary	Miller et al. 1973, 19
	water	0.56	4	NG	6.9	12-18			64	NOAEC LC47 (reduced growth, food consumption, survival)	A/S/U secondary	
(6.6 g)	water	10.5	4	NG	7.7	3-8			56	(LT50 = 114 days)	A/S/U secondary	
	water	1.05	4	NG	7.7	3-8			56	NOAEC	A/S/U secondary	

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.1 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	Test Conditions pH	Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish Pike, Esox lucius L. (freshly fertilized eggs)	water	0.1 1.0 10	4		6.9	14±0.5		94-102		- inhibition of egg development (approx. 23% inhibition at all concentrations), dose related mortality, inhibition of growth of subsequent fry, approx. 100% mortality in hatched fry after 96H egg exposure to 10 ng/L (post-exposure observation period not given)	A/R/U secondary	Helder 1980a
Guppy, Poecilia reticulatus (9-40 mm)	water	100 1000 10000	5			20				fin necrosis in 10 d.,100% mortality in 37d, smaller fish more sensitive	A/S/U secondary	Norris and Miller 1974
Guppy, Poecilia reticulatus (8-12 mm)	water	0.1	1		6.9	21-29	-	-	64	LOAEL (fin necrosis)	A/S/U secondary	Miller et al. 1979
		0.01	1		6.9	21-29	-	-	64	NOAEC (after 42d for fin necrosis)		

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.1 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	Test Conditions pH	Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Fathead minnow, Pimephales promelas (juveniles, 0.5-1.0g)	water	1.7	28	25400 (dry wt. basis)	6.8-7.7	21-23	4.5-8.3	80	110	28D-LC50 (steady state fish concn. not reached)	C/R/U secondary	Adams et al. 1986
(1-2g)	water	7.1	1-4		6.8-7.7	21-23	4.5-8.3	80	110	LOAEC (mortality)	A/S/U secondary	
(1-2g)	water	0.7	1-4		6.8-7.7	21-23	4.5-8.3	80	110	NOAEC (150d observation period)	A/S/U secondary	
Rainbow trout, Salmo gairdneri (eggs)	water	0.1	4			10± 0.5				delayed embryo development and growth	C/R/U secondary	Helder 1980b, 1981
	water	100	4							100% mortality	C/R/U secondary	
(yolk sac fry)	water	10	4							100% mortality	C/R/U secondary	
(juveniles, mean 0.85g)	water	100	4							100% mortality within 27 days	C/R/U secondary	
(eggs-fry)	water	10	4							growth inhibition over 72d period following fertilization	C/R/U secondary	
(newly hatched larvae)	water	1	4							inhibition of growth over 72d observation period	C/R/U secondary	

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.1 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	pH	Test Conditions Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Rainbow trout, Salmo gairdneri (fry, 0.38 ± 0.09g)	water	.038	28	980	7.7	12±1	65-85	88	153	reduced growth (NOAEC < 0.038 ng/L)	C/R/M secondary	Mehrle et al. 1988
	water	0.79	28	NG	7.7	12±1	65-85	88	153	no observed mortality but reduced growth and behavioural stress during exposure period; significant mortality during 28d depvration period at both concn.	C/R/M seconday	
	water	.176	28	4520	7.7	12±1	65-85	88	153	50% mortality in 28d	C/R/M secondary	
	water	.382	28	10950	7.7	12±1	65-85	88	153	73% mortality in 28d	C/R/M secondary	
	water	.789	28	15410 (at day 21)	7.7	12±1	65-85	88	153	85% mortality in 28d	C/R/M secondary	

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.1 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	Test Conditions pH	Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Carp	water	0.10	71	approx. 2000						weight loss and other chronic effects	C/-/U? secondary	Cook et al. 1986
Molluscs												
Snail, Physa sp. (juvenile and adult)	water	200 (initial)	48	NG	6.9	23-27			64	reduced reproduction but not significant based on variability in replicates	C/S/U secondary	Miller et al. 1973
Oligochaete Worms Paranais sp.	water	200 (initial)	55	NG						significant reduction in reproduction	C/S/U	Miller et al. 1973

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.1 (CONTINUED)

Compound: 2,3,7,8-T_4CDF

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	pH	Test Conditions Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Rainbow trout, Salmo gairdneri	water	0.41	28		7.7	12±1	65-85%			-NOEC (growth)	C/R/M primary	Mehrle et al. 1988
		0.90	28		7.7	12±1	65-85%			-growth effects		
		1.79	28		7.7	12±1	65-85%			-no observed mortality during 28d exposure or 28d depuration period; signif. growth decrease		
		3.93	28		7.7	12±1	65-85%			-signific. mortality within 14d		
		8.78	28		7.7	12±1	65-85%	88	153	-signific. mortality within 14d.		

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.2 - ANCILLARY AQUATIC TOXICITY DATA

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	pH	Test Conditions: Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Rainbow trout, *Salmo gairdneri*	oral	6.3 ug/kg b.w	28			11-13				fin necrosis in 14d; some deaths in 33d	C/F/U ancillary	Miller *et al*. 1973
(juveniles)	oral	.0063 ug/kg b.w	28							NOAEL (33d observation period)	C/F/U ancillary	
Rainbow trout, *Salmo gairdneri*	oral	between 2.3 ppm and 2.3 ppb diet	105	1380000-1573 (one sample only for each dietary level)		15				NOAEL (mortality, growth, feeding, fin erosion)	C/F/U ancillary	Hawkes and Norris 1977
(7.78±0.97 cm)	oral	2.3 ppm diet	105	1380000 (one sample only)						LT50 = 61D (oral exposure to 2.3 ppm diet)	C/F/U ancillary	
Rainbow trout, *Salmo gairdneri*	ip	single injection 1.2 ug/kg b.w.				12				elevated liver cytochrome P-450 content	A/-/- ancillary	Vodicinik *et al*. 1981
(immature) 100-200g												
Rainbow trout, *Salmo gairdneri*	food	494	91	250						no effect	ancillary	Kleeman *et al*. 1986a
Yellow perch, *Perca flavescens* (5-10 g)	food	494 ng/kg	91	143						no effect (growth, fin necrosis, hemorrhage, lethality)	C/F/M ancillary	Kleeman *et al*. 1986b
Carp, *Cyprinus carpio* (10g)	sediment	39	55	7.5		25±1				no effect (water concn = ND at DL = 0.3 pg/L)	C/F/M ancillary	Kuehl *et al*. 1987a

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE 8.2 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	pH	Test Conditions Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Fish												
Rainbow trout, Salmo gairdneri (35± 10g)*	ip	10000	single injection			16±2				LD50 (80 D post-treatment observation)	A/F/-ancillary	Kleeman et al. 1988 *(fish weights reported as received, 4-W acclimation period used)
Yellow perch, Perca flavescens (40 ± 6g)*	ip	3000	single injection			16±2				LD50 (80 D post-treatment observation)		
Carp, Cyprinus carpio (20± 7g)*	ip	3000	single injection			16±2				LD50 (80 D post-treatment observation)		
Bluegill, Leponis macrochirus (30 ± 4g)*	ip	16000	single injection			16±2				LD50 (80 D post-treatment observation)		
Largemouth bass, Micropterus salmoides (7 ± 2g)*	ip	11000	single injection			16±2				LD50 (80 D post-treatment observation)		
Bullhead, Ictalurus melas (6 ± 2g)*	ip	5000	single injection			16±2				LD50 (80 D post-treatment observation)		

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.2 (CONTINUED)

Compound: 2,3,7,8-T_4CDD

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	Test Conditions pH	Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Amphibians												
Bullfrog, Rana catesbeiara (tadpole)	ip	500 ug/kg b.w.	single injection							no effect through metamorphasis	ancillary	Neal et al. 1979
Bullfrog, Rana catesbeiara (adult)	ip	500 ug/kg b.w.	single injection							no effect 35 day post exposure observation	ancillary	
Arthropods												
Mosquito, Aedes aegypti (larvae)	water	200 (initial)	17	NG	6.9	23-27	-	-	64	no observed effects (39 day observation period) (only conc. tested).	C/S/U ancillary	Miller et al. 1973
Waterflea, Daphnia magna (adult)	water	2.4-4.2	32	(17.1 ppb) 17100 max.						no effect (feeding, growth, reproductive activity) (water concn. derived from 0.1 ppm 14C-T_4CDD treated soil)	C/S/M ancillary	Yochim et al. 1978
Waterflea, Daphnia magna (<1,7,21d)	water	0.2-1030	2	ND	6.8-7.7	21-23	4.5-8.3	80	110	no effects at any exposure concentration (9 day observation period)	A/S/M ancillary	Adams et al. 1986

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

TABLE B.2 (CONTINUED)

Species	Route	Dose or Conc. (ng/kg or ng/L)	Expos. Time (days)	Conc. in whole organism (ng/kg)	pH	Test Conditions Temp. (°C)	D.O. (mg/L)	Alk. (mg/L)	Hardn. (mg/L)	Effect	Data	Reference
Compound: 2,3,7,8-T_4CDD												
Molluscs												
Snail, Helosoma sp. (adult)	water	2.4-4.2	32	9700						no effect (feeding, growth, reproductive activity) observed (water concn. derived from 0.1 ppm 14C-T_4CDD treated soil)	C/S/M ancillary	Yochim et al. 1978
Compound: 1,2,3,4,7,8-H_6CDD												
Fish												
Rainbow trout, Salmo gairdneri (fry, 0.1-0.3g)	water	10	5	3500		10	7-8			no effect noted during 48h depvration period	A/F/M ancillary	Muir et al. 1985b, 1988
Rainbow trout, Salmo gairdneri	food	100000 ng/kg	30	7000		10	7-8			possible growth reduction but not significant.	ancillary	Muir et al. 1988
Compound: 1,2,3,4,6,7,8-H_7CDD												
Fish												
Rainbow trout, Salmo gairdneri (fry, 0.1-0.3 g)	water	11	5	2500		10	7-8			no effect	A/F/M ancillary	Muir et al. 1985b, 1988
Rainbow trout, Salmo gairdneri	food	10000	30	2000		10	7-8			no effect	C/F/M ancillary	Muir et al. 1988

DATA CODES

A = Acute M = Measured
C = Chronic U = Unmeasured
R = Renewal S = Static
F = Flow-through

appendix c

Form C: Guideline Derivation Graph

2,3,7,8 – T_4CDD

Information considered	Species and life stage	Effect and exposure time	Concentration units: (pg/L) 0.1 1.0 10 100 1000 10000	Ref. No.
Acute data:				
Fish	Pike, egg	4d – growth	○	16
	Guppy,(9 – 40mm)	1d – LOEL,fin necrosis	○	33
Invertebrates	Coho salmon, juveniles	4d – LC_{50}	○	32
Chronic data:				
Fish	Rainbow trout, fry	28d – growth	*	31
	Fathead, juveniles	28d – LC_{50}	○	3
	Catfish, fingerlings	20d – LC_{50}	○	61
Invertebrates	Worm, Paranais sp	49d – reprod.	○	32
Plants				
Preliminary Organoleptic Guideline				
Preliminary BCF Guideline			△ ▲	
PWQG			⊕	
Objectives of other agencies				

Legend

- Primary ●
- Secondary ○
- PWQG ⊕
- Other Agencies □
- Lowest Effect *
- BCF Human Health ▲
- BCF Wildlife Protection △

Form C: Guideline Drivation Graph

2,3,7,8 – T_4 CDF

Information considered	Species and life stage	Effect and exposure time	Concentration units: (pg/L) 0.1 1.0 10 100 1000 10000	Ref. No.
Acute data: Fish				31
Invertebrates				
Chronic data: Fish	Rainbow trout fry	28d – growth	*	
Invertebrates				
Plants				
Preliminary Organoleptic Guideline				
Preliminary BCF Guideline				
PWQG			⊕	
Objectives of other agencies				

Legend

- Primary ●
- Secondary ○
- PWQG ⊕
- Other Agencies □
- Lowest Effect *
- BCF Human Health ▲
- BCF Wildlife Protection △

appendix d

Form C: Uncertainty Factor Worksheet

Chemical name: 2,3,7,8 – T_4CDD

Concentration units: ng/L

chronic toxicity data:

	Fish Species			Invert. Family		Plant
Effect Level (1)	0.038	1.7	2.4	200		
Reference Number	31	3	61	32		
Primary/Secondary/Simulated Data (2)	2°	2°	2°	2°		
Toxicity End-point Code (3)	G	M	M	R		
Calibration Factor	0.5	0.5	0.5	0.5		

acute toxicity data:

	Fish Family			Invert.Family	
Effect Level	0.1	0.1	5.6		
Reference Number	16	33	32		
Primary/Secondary/Simulated Data	2°	2°	2°		
Toxicity End-point Code	G	O	M		
Calibration Factor	0.8	0.8	0.8		

Calculation of final uncertainty factor :

Baseline factor x Calibration factors (max number = 11)

10000 X 0.5 X 0.5 X 0.5 X 0.5 X 0.8 X 0.8 X 0.8 X ___ X ___ X ___ X ___

= 320 Final uncertainty factor, rounded to nearest whole number.

(1) Express all concentrations in the same units.

(2) 1° = primary; 2° = secondary; 3° = simulated by ACR and /or QSAR

(3) Toxicity end-point codes: L = lethality; G = growth; R = reproduction; B = behavioral; O = other

Form C: Uncertainty Factor Worksheet

Chemical name: 2,3,7,8 – T_4 CDF

Concentration units: ng|L

chronic toxicity data:

	Fish Species			Invert. Family		Plant
Effect Level (1)	0.90					
Reference Number	31					
Primary/Secondary/Simulated Data (2)	1°					
Toxicity End-point Code (3)	G					
Calibration Factor	0.4					

acute toxicity data:

	Fish Family			Invert.Family	
Effect Level					
Reference Number					
Primary/Secondary/Simulated Data					
Toxicity End-point Code					
Calibration Factor					

Calculation of final uncertainty factor :

Baseline factor x Calibration factors (max number = 11)

10000 x 0.4 x [ ] x [ ] x [ ] x [ ] x [ ] x [ ] x [ ] x [ ] x [ ] x [ ] x [ ]

= 4000 Final uncertainty factor, rounded to nearest whole number.

(1) Express all concentrations in the same units.

(2) 1^o = primary; 2^o = secondary; 3^o = simulated by ACR and /or QSAR

(3) Toxicity end-point codes: L = lethality; G = growth; R = reproduction; B = behavioral; O = other

7540-1021

MINISTRY OF THE ENVIRONMENT

JAN 12 [illegible]

HAZARDOUS CONTAMINANTS BRANCH

SEND TO: Brendan Birmingham
Hazardous Contaminants Branch.

FROM: Conrad deBarros. DEPT. Water Resources Branch DATE Jan 12/90

SUBJECT: PWQO - Dioxins & Furans.

MacLaren Plansearch has completed their contract and supplied the attached final draft document.

Unfortunately it does not conform to the PWQO process that was circulated widely for peer review. I will therefore be making some minor modifications to ensure consistency with our PWQO process. When these changes have been made I will

REPLY: circulate a copy of the revised draft document to all members of the ad hoc committee. and communicating the changes to the HCDC.

Conrad deBarros.

(Not MacLarens fault PWQO process was finalized after contract initiation).

REPLY FROM

REPLY DATE

TO WRITE: HANDWRITE OR TYPE. REMOVE AND RETAIN YELLOW COPY FORWARD BALANCE OF SET

TO REPLY: WRITE REPLY IN BOTTOM AREA. SNAP SET APART

Ontario

FOLD AT MARKS FOR USE IN #9 OR # 10 WINDOW ENVELOPE

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TD
370
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1989

Development of provincial water quality guidelines for chlorinated dioxins and furans. by Mart Lupp and Lynn S. McCarty.

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quality guidelines for chlorinate
dioxins and furans. by Mart
Lupp and Lynn S. McCarty.

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